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The neuromuscular demands of altering foot progression angle during gait in asymptomatic individuals and those with knee osteoarthritis

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SUMMARY

Objectives: To determine the immediate effects of a toe-out foot progression angle modification during gait on the major lower limb muscle activation characteristics and to establish whether asymptomatic individuals and those with moderate knee OA have similar responses.

Design: Seventeen patients with knee OA and 20 asymptomatic control subjects participated. Informed consent was obtained. Electromyographic (EMG) recordings were acquired from the lateral and medial gastrocnemii, vastus lateralis, vastus medialis, rectus femoris and the lateral and medial hamstrings during neutral and toe-out walking conditions. The EMG waveforms were amplitude normalized to maximal voluntary isometric contractions and time normalized to the gait cycle. Principal component analysis extracted principal waveform features. Analysis of variance models tested for main effects and interactions. Bonferroni *post hoc* testing was employed ($\alpha = 0.05$).

Results: Both groups altered foot progression angle by approximately 15° during toe-out walking ($P < 0.05$). A shift in gastrocnemius activation towards later stance ($P < 0.05$) and increased magnitude and duration of quadriceps activation ($P < 0.05$) was found. A differential activation occurred in the overall magnitude and principal shape of the lateral and medial hamstring musculature in the asymptomatic group only ($P < 0.05$). Significant group differences were shown in each muscle analysis ($P < 0.05$).

Conclusion: Neuromuscular demands of adopting a toe-out gait differ from a neutral foot progression angle. Demands also differ between asymptomatic controls and patients with moderate knee OA. These findings have relevance for altered joint loading and changes in metabolic cost of this gait modification in individuals with knee OA.

Introduction

Knee osteoarthritis (OA) continues to be a leading cause of ambulatory disability in older adults[1–3]. Although many joints may be implicated, the prevalence of symptomatic knee OA appears to be most frequent[4–6]. This presents an escalating burden, both on the individual and health care systems[7,8]. Findings of instability, reduced knee joint proprioceptive acuity and knee extensor muscle weakness in those with this disease suggest the integration of both sensory and motor systems is impaired[9–14].

Recent studies have investigated lower extremity neuromuscular function associated with self-selected gait in individuals with knee OA. Increased agonist/antagonist co-activation strategies and differential recruitment of musculature with medial and lateral orientations have been reported[10,15–18]. Current findings suggest that individuals with mild to moderate medial compartment knee OA selectively recruit their lateral musculature (biceps femoris, vastus lateralis (VL)) during early and mid-stance with greater amplitudes and for longer duration than medial musculature, hypothesized to assist in unloading the medial tibio-femoral joint compartment[15,16]. Greater levels of co-activity found between medial gastrocnemius (MG) and vastus medialis (VM) during pre and early stance is a mechanism thought to increase stiffness in the medial compartment[10]. These alterations in knee joint muscle activation contribute to increased energy expenditure and different moments of force about the knee in both the sagittal and frontal

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plane; therefore understanding how these activation patterns are altered with therapeutic techniques used to manage knee OA is essential[19–21].

Adopting a toe-out foot progression angle during gait has been examined as a gait modification strategy for individuals with knee OA based on evaluating the knee adduction moment during walking[15,22,23]. A toe-out position has been associated with reduced net external knee adduction moment magnitude during late stance, providing indirect evidence of an attenuated tibio-femoral loading environment[15,23–25]. In contrast, only one recent study has addressed whether the neuromuscular demands are influenced with toe-out gait. Lynn and Costigan[15] found that levels of medial and lateral hamstring activation during stance phase were altered with toe-out gait in a cohort of 12 participants diagnosed with knee OA. While this result suggests a change in the hamstring musculature activation balance, the effects of toe-out gait on other muscles crossing the knee joint such as the quadriceps and gastrocnemius activation that are altered in the presence of knee OA have not been evaluated[10,16].

While most studies evaluate electromyographic (EMG) amplitude characteristics, temporal waveform patterns provide information on the dynamically changing amplitude and shape throughout the gait cycle[10,15–17,26]. To our knowledge, the amplitude and temporal activation profiles of both medial and lateral orientated quadriceps, hamstrings and gastrocnemii musculature have not been investigated in the context of foot progression angle modifications during gait in asymptomatic controls or individuals with knee OA. This information can shed light on the tibio-femoral joint loading environment and the metabolic cost of gait.

The purpose of this study was twofold: 1) to determine the immediate effects of a toe-out foot progression angle modification on knee joint muscle activation characteristics and 2) to determine whether asymptomatic individuals and those with moderate knee OA have similar neuromuscular responses. We hypothesized that adopting a toe-out foot progression angle alters the amplitude and temporal neuromuscular characteristics throughout the gait cycle and these alterations will differ between those with and those without knee OA.

Method

Subjects

Asymptomatic controls ($n = 20$) were recruited from the general community and presented with no known symptoms of degenerative joint disease in the lower extremity. Participants with unilateral symptomatic knee OA ($n = 17$) were recruited from the caseload of one high volume orthopaedic surgeon (WDS). Standard anterior/posterior radiographs confirmed predominant medial compartment radiographic disease presence and were scored using the Kellgren Lawrence global scoring algorithm[27]. The knee OA group were required to safely walk one city block, reciprocally ascend and descend 10 stairs and jog 5 m[16]. In addition to above, inclusion criteria for both groups included: age of 35 years or older, no cardiovascular/respiratory disease or neurological disorders present, no lower limb surgery, fracture or injury other than a sprain or strain (greater than 1-year). Gait analysis was completed at the Dynamics of Human Movement Laboratory, Dalhousie University, Halifax, Nova Scotia. Written informed consent using procedures approved by the local institutional ethics review committee was attained.

EMG gait analysis

The lower extremity tested during gait for the asymptomatic group was randomly assigned and for the knee OA group, was the affected lower extremity. Standard skin preparation (light shave and vigorous clean with 70% alcohol wipes) and placement of surface electrodes in a bipolar configuration (Ag/AgCl, 10 mm diameter, 20 mm interelectrode distance) over the VM, VL, rectus femoris (RF), semitendinosis/membranosis (MH), biceps femoris (LH), lateral gastrocnemius (LG) and MG were performed by an experienced orthopaedic physiotherapist (DJR)[16]. Muscle palpation and an isometric contractions series were used to validate electrode placement[28] and for setting appropriate gain adjustments. Myoelectric signals were amplified using an AMT-8 (Bortec, Inc., Calgary, Alberta, Canada), eight-channel EMG system (input impedance: $\sim 10\ G\Omega$, Common Mode Rejection Ratio (CMRR): 115 dB at 60 Hz, band-pass (10–1000 Hz)).

Three-dimensional motion capture procedures have been previously described[22]. Infrared emitting diode (IRED) skin surface markers were affixed to the lateral aspect of the shoulder, greater trochanter, lateral epicondyle and lateral malleolus with three non-collinear IRED marker clusters placed on the thigh, lower leg and foot. Skin surface markers, electrodes, pre-amps and lead wires were secured with adhesive tape and nylon stocking.

Subjects were instructed to walk at their self-selected velocity along a 6-m walkway. After three familiarization trials, seven walking trials were collected. Their neutral angle was determined from the computer analysis. Using a standard goniometer, a template for foot placement of at least 10° greater than neutral was marked at the beginning of the walkway. After three familiarization trials in which subjects walked with at least an additional 10° of toe-out angle from their neutral angle, seven trials of toe-out walking were recorded. Photoelectric timers, positioned at known distances on the walkway, monitored walking velocity. Subjects were required to walk within $\pm$ 0.1 m/s of the "neutral" walking condition velocity for toe-out trials.

Following the walking trials, baseline muscle activity was recorded while subjects lay relaxed in supine lying. They then completed eight standardized exercises to elicit maximal voluntary isometric contraction (MVIC) efforts for EMG normalization purposes[16]. Exercises included 1) knee extension from sitting (knee position was 45° flexion) 2) knee extension/hip flexion from sitting (knee position was 45° flexion) 3) knee flexion from sitting (knee position was 55° flexion) 4) knee extension from supine lying (knee position was 15° flexion) 5) knee flexion from supine lying (knee position was 15° flexion) 6) ankle plantar flexion from long sitting (neutral ankle position) 7) standing unilateral heel raise and 8) knee flexion from prone lying (knee position was 55° flexion). Following at least one practice and warm-up contraction two, 3-s maximal isometric contractions were completed for each exercise. A 60-s rest period separated each contraction, and standardized verbal encouragement was given[16]. All EMG signals were analogue to digital converted at 1000 Hz (16 bit, $\pm$2 V) and stored for processing.

Lower extremity motion, captured at 100 Hz using two opto-electronic motion analysis sensors (Optotrak™, Northern Digital Inc., Waterloo, ON) and ground reaction forces, captured at 1000 Hz using a single force plate (AMTI™, Advanced Mechanical Technology Incorporation, Newton, MA, USA) embedded in the walkway were used to identify heel strike and toe off events for the time normalization of the electromyogram[16].

Data processing

Raw EMG data was processed through custom MatLab™ Ver 7.1 programs (The Mathworks Inc., Natick, Massachusetts, USA). All EMG signals were corrected for bias and converted to micro-volts, full wave rectified and low pass filtered (Butterworth, fourth order, Fc-6 Hz). After visual inspection for artefact, each EMG waveform was time normalized to represent 100% of the gait cycle and

amplitude normalized to MVIC. For amplitude normalization, a 100 ms moving window algorithm identified the maximal EMG amplitude for each muscle across all eight MVIC exercises[16]. The foot progression angle was derived as the angle between the line of progression and the long axis of the foot (base of second metatarsal to center of calcaneous defined as virtual points with respect to the marker triad affixed to the foot)[22].

Analysis

Ensemble average EMG waveforms were determined for each subject corresponding to each muscle and each condition[29]. Principal component analysis (PCA) was used to capture amplitude and temporal features in the gait waveforms using custom MatLab™ Ver.7.1 programs (The Mathworks Inc., Natick, Massachusetts, USA). Three separate PCA were performed, one for each muscle grouping. Time-normalized waveforms (101 points) from the corresponding individual muscles, the two walking conditions, and the two groups, formed three matrices (X); 1) gastrocnemius musculature (148 waveforms), 2) quadriceps musculature (222 waveforms) and 3) the hamstring musculature (148 waveforms). An eigenvector decomposition of the cross product matrix ($[S] = [X^T] \times [X]$) was performed, using standard notation $U'SU = L$, yielding the predominant orthonormal components called eigenvectors[16,30]. These eigenvectors are the principal patterns. The principal patterns explaining the greatest percent of variation in the waveforms were retained and referred to as PP1, PP2 etc.[16]. Principal pattern scores (*PP-scores*) were computed for individual gait waveforms in each separate analysis ($PP\text{-}score = [X] \times {}^*[U]$). The *PP-score* is a weighting coefficient for each principal pattern related to each measured EMG waveform. The measured EMG waveform can be accurately reconstructed by the linear combination of the principal patterns multiplied by the corresponding *PP-scores*. How many of principal patterns needed to pick up the salient information in the measured waveform was estimated by the percent trace[16,26]. The individual *PP-scores* for each muscle within a grouping were scored against a common principal pattern thus allowing for statistical hypothesis testing to compare the characteristics of the waveforms between muscle sites within a muscle group and between conditions[16,26]. For qualitative comparisons, group ensemble average profiles were calculated for each muscle and for each condition[16].

Statistical analysis

Student *t* tests were used to test for significant differences between the two groups for age, mass, height, body mass index (BMI). Two-factor (group, condition) Analysis of Variance (ANOVA) model tested for differences in self-selected walking velocity, foot progression angle and stride length characteristics. The main hypotheses were tested using a three-factor mixed model ANOVA, for group (asymptomatic or knee OA), with repeated measures on condition (neutral or toe-out foot progression angles) and muscle main effects and all interactions against relevant *PP-scores*. Separate statistical analyses were completed for each of the three muscle groupings. Bonferroni *post hoc* analysis was performed on all significant findings ($\alpha = 0.05$). Normality and equal variance were tested utilizing a Kolmogorov–Smirnov and a Levene's test respectively ($\alpha = 0.05$). Statistical procedures were completed on Minitab™ Ver.15 (Minitab Inc. State College, PA, USA).

Results

The knee OA group had greater mass and greater BMI than asymptomatic individuals and were significantly older ($P < 0.05$) (Table I). Forty-one percent of the knee OA group were females whereas females made up 65% of the asymptomatic group. Kellgren Lawrence scores for the OA groups included one subject with grade I, seven with grade II, five with grade III and two with grade IV radiographic changes. Two subjects were not scored. While walking velocity was greater in the asymptomatic group ($P < 0.05$), no differences between toe-out and neutral conditions existed for walking velocity ($P > 0.05$). Foot progression angle was 15° greater for toe-out gait which was statistically significant ($P < 0.05$) (Table I).

Group ensemble-averaged profiles for two walking conditions for the gastrocnemius, hamstrings and quadriceps along with the principal patterns are in Figs. 1–3. Also depicted is the mean of five measured EMG waveforms associated with high and low *PP-scores* to assist with interpretation. *PP-scores* for each group, condition and muscle are found in Tables II and III with statistical results on Table IV.

Three principal patterns [Fig. 1(C)] captured 97% of the waveform variance in gastrocnemius activation. Significant condition ($P < 0.05$) effects for *PP2-scores* and significant ($P < 0.05$) group and muscle main effects for *PP3-scores* were found (Table IV). Neutral gait *PP2-scores* were greater than toe-out gait *PP2-scores* ($P < 0.05$). High *PP2-scores* indicate a phase shift in activity to earlier in stance phase [Fig. 1(E)]. The asymptomatic group had greater *PP3-scores* scores than the knee OA group ($P < 0.05$) indicating a greater difference in amplitude between early and late stance. The MG had greater *PP3-scores* than LG ($P < 0.05$).

Group ensemble-averaged LH and MH waveforms are in Fig. 2. Ninety four percent of the waveform variance were captured with three principal patterns [Fig. 2(C)]. Unequal variance and non-normality were apparent in *PP-scores* for the hamstrings ($P < 0.05$). A three-way group by muscle by condition interaction was found for *PP1-scores* (Table IV). Lower LH *PP1-scores* were found in asymptomatic group compared to knee OA group for both neutral and toe-out gait ($P < 0.05$). LH *PP1-scores* were greater than MH *PP1-scores* during both neutral and toe-out gait ($P < 0.05$) in the knee OA group and only during the toe-out condition for asymptomatic group ($P < 0.05$). In the asymptomatic group only, LH *PP1-scores* were lower during neutral gait compared to toe-out gait ($P < 0.05$), and the MH *PP1-scores* were higher during neutral gait than during toe-out gait ($P < 0.05$). High *PP1-score* indicates higher overall activation amplitude. A significant group by muscle interaction was found for *PP2-scores* and group by condition interaction

Table I
Mean and Standard Deviation (SD) for group demographics and gait characteristics

	Asymptomatic	Moderate knee OA	*P*-value
N	20	17	
# Females	13	7	
Mass (kg)	75.5 (16.1)	88.8 (19.7)	**$P = 0.031$**
Height (m)	1.70 (0.07)	1.72 (0.09)	$P = 0.424$
BMI (kg/m^2)	25.9 (4.8)	29.8 (6.5)	**$P = 0.044$**
Age (years)	46.5 (7.0)	56.0 (8.8)	**$P = 0.0007$**
Gait characteristics			
Velocity (m/s)			
Neutral	1.44 (0.14)	1.25 (0.15)	**Group $P = 0.000$**
Toe out	1.46 (0.14)	1.26 (0.16)	Cond $P = 0.167$
Stride length (m)			
Neutral	1.50 (0.11)	1.39 (0.17)	**Group $P = 0.02$**
Toe out	1.52 (0.12)	1.39 (0.17)	Cond $P = 0.225$
Foot progression angle (degrees)			
Neutral	4.9 (4.7)	6.6 (7.3)	Group $P = 0.625$
Toe out	21.6 (5.0)	21.7 (7.2)	**Cond $P = 0.000$**

Significant findings are in bold.

Table II
Mean and Standard Deviation (SD) *PP-scores* for each group and condition for gastrocnemius and hamstring musculature

Group	Asymptomatic				Moderate knee OA			
Muscle	Lateral		Medial		Lateral		Medial	
Condition	Neutral	TO	Neutral	TO	Neutral	TO	Neutral	TO
Gastrocnemius								
PP1-scores	223.9 (82.8)	208.1 (76.1)	242.8 (95.4)	258.4 (104.6)	194.0 (71.1)	185.0 (72.5)	210.7 (111.0)	205.7 (81.7)
PP2-scores	−15.5 (50.6)	−21.6 (35.5)	11.7 (50.4)	0.1 (54.6)	2.8 (42.8)	−9.1 (52.2)	9.4 (48.0)	−11.4 (41.5)
PP3-scores	−1.4 (34.7)	−2.7 (32.3)	15.6 (33.3)	23.4 (36.7)	−25.9 (41.3)	−30.0 (31.9)	−9.6 (39.4)	−5.7 (38.4)
Hamstring								
PP1-scores	81.7 (30.9)	95.7 (31.0)	90.7 (33.4)	78.2 (31.3)	148.9 (61.4)	146.3 (58.0)	94.2 (32.6)	87.1 (31.1)
PP2-scores	−21.7 (25.7)	−21.3 (26.6)	−21.9 (22.5)	−17.1 (17.8)	22.5 (47.6)	23.7 (45.6)	−0.7 (23.0)	4.7 (18.5)
PP3-scores	−4.6 (11.7)	−3.7 (10.3)	−8.7 (18.0)	−6.5 (11.0)	4.13 (34.8)	−5.5 (36.7)	6.1 (15.4)	2.6 (16.6)

Gastrocnemius: Lateral = LG, Medial = MG.
Hamstring: Lateral = Lateral hamstring, Medial = Medial hamstring.
Neutral = Neutral gait, TO = Toe-out foot progression angle.

for *PP3-scores* (Table IV). In the knee OA group, LH *PP2-scores* were greater than MH *PP2-scores* ($P < 0.05$). Asymptomatic LH and MH *PP2-scores* were lower than the respective muscle *PP2-scores* in the knee OA group ($P < 0.05$). High *PP2-scores* indicate prolonged activation and no burst of activity prior to heel strike. In the knee OA group, *PP3-scores* were lower for toe-out gait compared to neutral gait ($P < 0.05$). For neutral condition, *PP3-scores* of the asymptomatic group were lower than knee OA *PP3-scores* ($P < 0.05$). High *PP3-scores* capture high activation early in stance and late in swing.

For the quadriceps, three principal patterns captured 96% of the waveform variance [Fig. 3(D)]. Significant condition and muscle main effects were found for *PP1-scores* (Table IV). Neutral gait *PP1-scores* were lower than those found for toe-out gait ($P < 0.05$). VL and VM *PP1-scores* were not different from each other ($P > 0.05$) but greater than RF *PP1-scores* ($P < 0.05$). High *PP1-scores* indicate higher overall activation. Significant condition and muscle main effects were found for *PP2-scores* and *PP3-scores* as well as significant group effect for *PP3-scores* (Table IV). Neutral gait *PP2-scores* were greater than toe-out gait *PP2-scores* ($P < 0.05$). VL *PP2-scores* were greater than both the VM and RF *PP2-scores* ($P < 0.05$). VM *PP2-scores* were greater than RF *PP2-scores* ($P < 0.05$). High *PP2-scores* indicate reduced activity mid to late stance phase compared to early stance. Unequal variance and non-normality were apparent in *PP2-scores* ($P < 0.05$). Asymptomatic *PP3-scores* were greater than knee OA group *PP3-scores* ($P < 0.05$). Toe-out gait *PP3-scores* were lower than neutral gait *PP3-scores* ($P < 0.05$). VL and VM *PP3-scores* were greater than RF *PP3-scores* ($P < 0.05$) and VL *PP3-scores* were lower than VM *PP3-scores* ($P < 0.05$). High *PP3-scores* indicate a reduced mid-stance compared to late stance.

These results illustrate that amplitude and temporal differences were found between groups, muscles and conditions for the three muscle groupings. Significant interactions for the hamstrings indicate different responses to toe-out alterations for the knee OA group compared to asymptomatic controls.

Discussion

Muscle activation levels can alter the joint loading environment and metabolic costs during walking. Determining the effects of conservative management strategies, such as gait modifications, based on corresponding neuromuscular demands provides a better understanding of their potential value. Those with mild to moderate knee OA were chosen for this study as previous work reported an association between foot progression angle and alterations in net external knee adduction moment that was not apparent in the severe knee OA group[22]. Also, neuromuscular recruitment strategies are sensitive to disease severity where individuals with moderate OA have been shown to differ from both asymptomatic controls and individuals with severe degenerative change[16,31]. This study found that altering foot progression angle towards a toe-out position changed lower extremity neuromuscular activity during self-selected walking and more importantly, changes were not consistent between asymptomatic and knee OA groups. These data provide insight on the potential effect of toe-out gait on medial/lateral joint loading, joint stability and metabolic demands of ambulation[10,15,16,21].

Gastrocnemius musculature

Toe-out foot progression angle alters the biomechanics of both the knee and foot[25]. Thus, one would expect a concurrent altering of myoelectric activity of the bi-articulate gastrocnemius musculature given that gastrocnemius function, knee and hind foot mechanics are related[10,32]. As shown in Fig. 1; toe-out gait did not significantly alter the overall magnitude and shape of the gastrocnemii waveforms. However, PP2 and PP3 identified changes that were not apparent from investigating the overall magnitude (PP1). During toe-out gait, the shift in the waveform towards later stance (lower *PP2-scores*) in both groups suggests that gastrocnemius activity during mid-stance, when the muscle is generating a plantar

Table III
Mean and Standard Deviation (SD) *PP-scores* for each group and condition for the quadriceps musculature

Group	Asymptomatic						Moderate knee OA					
Muscle	VL		VM		RF		VL		VM		RF	
Condition	Neutral	TO	Neutral	TO	Neutral	TO	Neutral	TO	Neutral	TO	Neutral	TO
Quadriceps												
PP1-score	118.5 (55.5)	130.2 (47.4)	122.9 (69.9)	132.2 (71.6)	68.2 (30.4)	85.3 (44.1)	144.8 (83.5)	166.0 (90.2)	142.5 (74.2)	156.0 (78.4)	92.8 (49.2)	112.0 (61.1)
PP2-score	16.6 (16.3)	15.9 (20.7)	17.2 (18.10	11.3 (24.4)	−9.7 (12.1)	−14.8 (18.5)	11.7 (34.6)	2.7 (37.8)	−1.4 (53.3)	−9.7 (55.6)	−8.2 (20.2)	−16.6 (20.5)
PP3-score	9.6 (22.6)	2.8 (20.1)	19.4 (27.7)	14.6 (29.4)	3.1 (6.2)	−6.4 (16.3)	−5.8 (28.7)	−18.8 (26.0)	4.3 (25.7)	−5.3 (26.2)	−9.1 (13.1)	−18.8 (17.4)

Neutral = Neutral gait, TO = Toe-out foot progression angle.

Table IV
P-values for *PP-score* main effects and interactions

	Principal pattern description	Group	Condition	Muscle	Group × condition	Group × muscle	Condition × muscle	Group × condition × muscle
Gastrocnemius								
PP1-scores	Overall magnitude	0.153	0.552	0.090	0.565	0.606	0.083	0.174
PP2-scores	Phase shift	0.723	**0.010**	0.125	0.422	0.195	0.210	0.766
PP3-scores	Early to late stance difference operator	**0.005**	0.729	**0.001**	0.717	0.920	0.070	0.917
Hamstring								
PP1-scores	Overall magnitude	0.004	0.417	0.000	0.279	0.001	0.000	**0.005**
PP2-scores	Prolonged activity and no late swing burst of activity	0.000	0.241	0.061	0.884	**0.026**	0.093	0.956
PP3-scores	High early stance and late swing vs mid-stance	0.180	0.181	0.815	**0.034**	0.228	0.135	0.328
Quadriceps								
PP1-scores	Overall magnitude	0.147	**0.000**	**0.000**	0.305	0.894	0.167	0.588
PP2-scores	Early to mid-late stance difference operator	0.241	**0.001**	**0.000**	0.169	0.111	0.557	0.361
PP3-scores	Mid to late stance difference operator	**0.007**	**0.000**	**0.000**	0.297	0.674	0.432	0.379

Significant findings are in bold.

flexion moment to control tibia rotation, was not required as early in stance as during neutral gait. This may effectively reduce the metabolic cost and duration of joint loading. Although it is difficult to directly compare principal patterns generated from different samples, PP1 and PP2 in this study are similar in shape to those found by Hubley-Kozey *et al.*[16] for asymptomatic controls and those with moderate knee OA. They reported group and muscle differences for *PP1-scores* and muscle differences for *PP2-scores*[16]. These differences were not found in the present study although similar trends were noted and lack of significance may be the result of the small sample size. In the current study, PP3 identified a pattern not previously reported where *PP3-scores* distinguished between groups (Fig. 1) but were unresponsive to the adoption of a toe-out foot progression angle. Low *PP3-scores* in the current study for the knee OA group elucidate an elevated activity during early stance compared to later stance. This strategy may increase stability in the tibio-femoral joint in early stance as suggested by Lewek *et al.*[10]. Lower peaks in late stance may be associated with the slower walking speed compared to asymptomatic controls[33]. Thus, while greater levels of gastrocnemius activation may benefit a knee OA population during early to mid-stance, the cost of greater muscle forces across the joint could become detrimental to the health of diseased articular cartilage and periarticular tissue[16,34]. Our findings support that alterations in the gastrocnemius musculature activation occur as a result of both toe-out gait and in the presence of knee OA.

Hamstring musculature

Higher LH activity in the knee OA group compared to both MH of the knee OA group and both hamstrings of the asymptomatic group (Fig. 2) is consistent with previous reports that individuals with knee OA present with elevated and prolonged LH activity to potentially reduce compressive loading in the medial

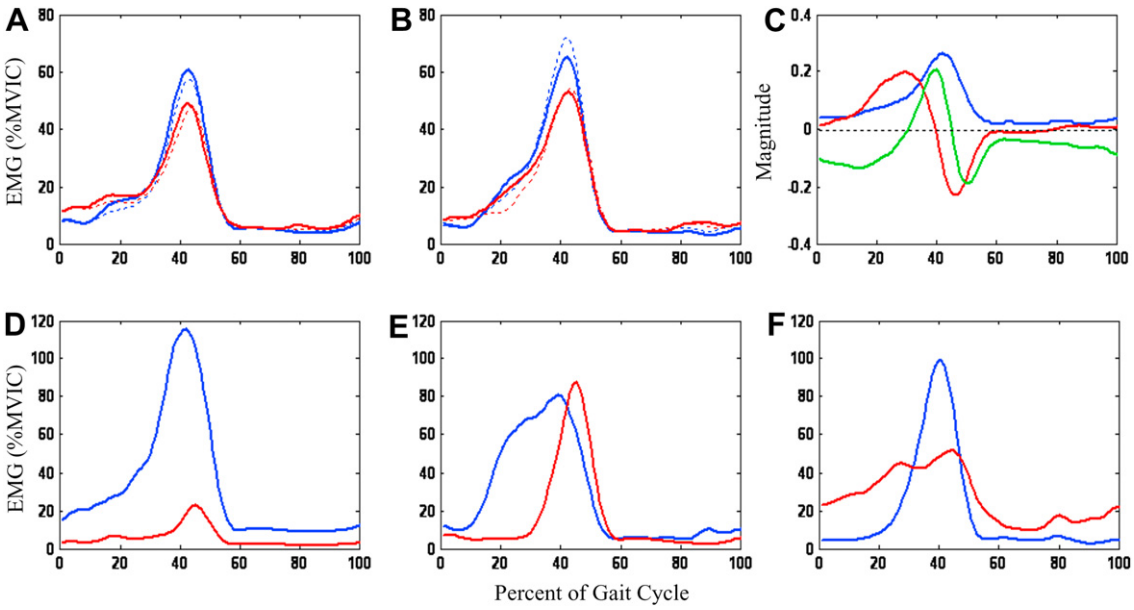

Fig. 1. Group ensemble average EMG profiles for A) LG and B) MG (solid-blue: neutral asymptomatic, solid-red: neutral knee OA, dotted-blue: toe-out asymptomatic, dotted-red: toe-out knee OA). C) Principal patterns (blue: PP1, red: PP2, green: PP3) and the mean of five waveforms corresponding to low (red) and high (blue) *PP-scores* for D) PP1, E) PP2 and F) PP3. PP1 captured the overall magnitude and general shape of the EMG waveforms associated with LG and MG, explaining 91% of the waveform variance. PP2 captured a phase shift in activation, explaining 3.8% of the waveform variance. PP3 acted as a difference operator between the early and late stance magnitudes, explaining 2.5% of the waveform variance.

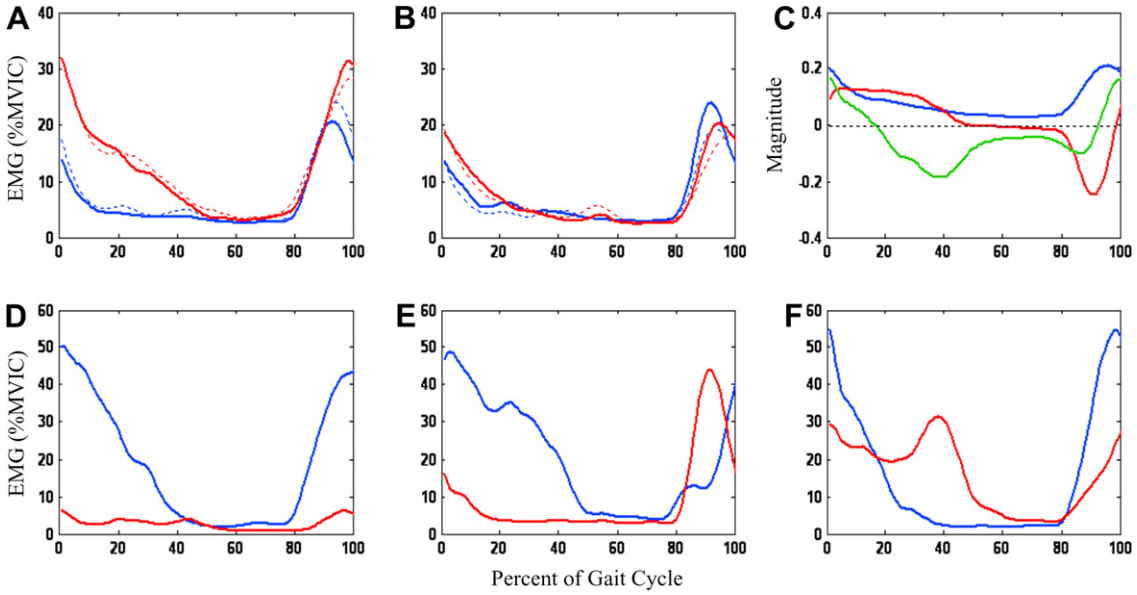

Fig. 2. Group ensemble average EMG profiles for A) LH and B) MH (solid-blue: neutral asymptomatic, solid-red: neutral knee OA, dotted-blue: toe-out asymptomatic, dotted-red: toe-out knee OA). C) Principal patterns (blue: PP1, red: PP2, green: PP3) and the mean of five waveforms corresponding to low (red) and high (blue) *PP-scores* for D) PP1, E) PP2 and F) PP3. PP1 captured the overall shape and magnitude, explaining 83% of the waveform variance. PP2 captured a prolonged elevated activation during early stance and the absent distinct burst of activity during late swing, explaining 8% of the waveform variance. PP3 explained 3% of the waveform variance and captured an elevated activation during early and late stance compared to mid-stance of the gait cycle.

compartment[15,16]. Adopting a toe-out gait did not significantly alter the overall amplitude (PP1) and prolonged (PP2) activity features for the LH or MH during the gait cycle in the knee OA group but did in the asymptomatic group. Higher LH overall amplitude during toe-out condition compared to MH and compared to LH during neutral gait along with lower MH activity during toe-out gait compared to neutral gait (Fig. 2) indicates that toe-out gait results in a differential recruitment strategy for asymptomatic controls. This strategy that biases LH activity and reduces medial site activity may unload the medial compartment in the asymptomatic group. Interestingly, these changes did not occur in the OA group. Increased amplitude (PP1) and prolonged activity (PP2) found for

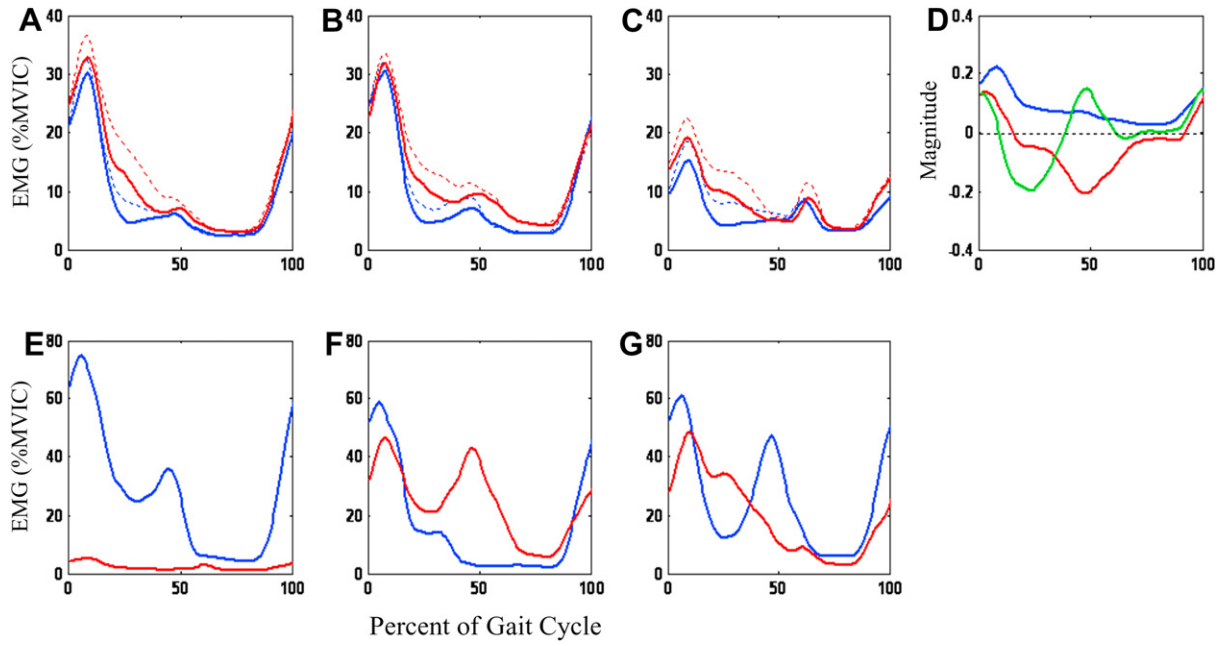

Fig. 3. Group ensemble average EMG profiles for A) VL, B) VM and C) RF (solid-blue: neutral asymptomatic, solid-red: neutral knee OA, dotted-blue: toe-out asymptomatic, dotted-red: toe-out knee OA). D) Principal patterns (blue: PP1, red: PP2, green: PP3) and the mean of five waveforms corresponding to low (red) and high (blue) *PP-scores* for D) PP1, E) PP2 and F) PP3. PP1 captured the overall magnitude and general shape of the electromyogram, explaining 88% of the waveform variance. PP2 explained 5% of the waveform variance and captured a reduced late stance magnitude in comparison to early stance. PP3 captured a reduced mid-stance magnitude compared to late stance and explained 3% of the waveform variance.

the hamstring muscles in the knee OA group was not altered by the toe-out condition. Thus, assuming that similar responses will be achieved in this patient group as with asymptomatic controls is not supported. *PP3-scores* suggest that in the knee OA group, an elevated activity was apparent in hamstrings during mid-stance for toe-out gait condition, potentially a result of heightened neuromuscular control required during the single leg stance phase. While Lynn and Costigan[15] found decreases in medial:lateral hamstring activation ratios for both asymptomatic and knee OA groups during stance phase of toe-out gait, the differences were not statistically significant for their asymptomatic group. Their ratio was a combination of decreased MH and increased LH activity over stance phase; a result that, in general, is similar to ours[15]. While group differences in hamstring activation levels are consistent with the literature, asymptomatic controls and those with knee OA did not respond to toe-out gait in a similar manner when features of the entire dynamic gait waveform were considered.

Quadriceps musculature

Adopting a toe-out foot progression angle increased the overall magnitude of the quadriceps musculature activation (PP1) and prolonged this activation (PP2 and PP3) during the stance phase of gait (Fig. 3). The lack of a significant interaction for *PP-scores* implies that this effect was equally apparent in both groups. As well, differential medial to lateral muscle site activations were not found in quadriceps musculature as VL, VM and RF were altered with the same direction and magnitude during toe-out gait. Higher amplitude quadriceps activation during toe-out gait has implications for its effectiveness. Prolonged activity of quadriceps both during neutral and toe-out gait is consistent with greater levels of co-activity that have been shown to occur in individuals with knee OA[16,17]. Increase in quadriceps activity provides indirect evidence of a higher metabolic cost associated with this gait modification compared to neutral gait. Also, individuals with knee OA present with elevated hamstring activity compared to asymptomatic individuals that was not altered with adoption of a toe-out gait. As a consequence, caution is needed if prescribing such modifications for those with knee OA as this modification may impact their fatigue levels during gait and subsequently, their ability to perform this functional task over longer periods.

These results provide insight into the value of the toe-out modification to reduce knee joint loading adding to our understanding derived from the net external moments measured through inverse dynamics. Essentially, the decrease in amplitude of the knee adduction moment during late stance is the most consistent finding reported[15,23–25]. The EMG findings indicate that this decrease in external moment is not associated with a consistent favorable change in activation levels of the major muscles to reduce muscle forces on the knee joint and more specifically on the medial compartment of the knee joint during stance.

Limitations need to be considered when interpreting these findings. Firstly, unequal variance and non-normality were apparent in the *PP-scores* for the hamstrings and quadriceps and are primarily the result of higher variability in the OA group. This higher variability in patient groups is common, but the current statistical results supported changes in the electromyogram and did not affect the overall interpretation of the findings with respect to the primary objectives. Group differences (Table I) were not included as covariates. While they potentially could explain some of the between group variability, they would be unable to account for the three-way interactions investigated in the present study. The between group differences were not the major aim of the study. In addition, the adjustment for baseline characteristics that are also features of the disease process may remove a portion of the between group variability. Finally, while we studied the immediate response of adopting a toe-out foot progression angle during gait, the question remains as to whether these adaptations would remain over long period changes. Longitudinal work is required to determine if neuromuscular changes are diminished or maintained with time and if clinically, relevant changes manifest in both the tibio-femoral loading environment and metabolic cost of walking.

Conclusion

A toe-out gait modification altered neuromuscular characteristics of the major muscle groups crossing the knee joint in both asymptomatic controls and those with moderate OA. Altered responses were consistent between groups for the gastrocnemius and quadriceps where a phase shift in gastrocnemius activity towards later stance and elevated and prolonged quadriceps activity occurred during toe-out gait. In contrast, toe-out gait altered hamstring activation characteristics differently between groups and muscle sites. The findings are relevant for establishing guidelines for implementing this gait modification in knee OA conservative management practices with respect to joint loading and metabolic cost. Where previous work has focused on gait biomechanics, these EMG results provide additional understanding of this gait modification strategy in individuals with knee OA.

Conflict of interest

The authors acknowledge that there are no conflicts of interest pertaining to this manuscript.

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